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## From the international space station to the clinic: how prolonged unloading may disrupt lumbar spine stability

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### Abstract

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#### BACKGROUND CONTEXT:

Prolonged microgravity exposure is associated with localized low back pain and an elevated risk of post-flight disc herniation. Although the mechanisms by which microgravity impairs the spine are unclear, they should be foundational for developing in-flight countermeasures for maintaining astronaut spine health. Because human spine anatomy has adapted to upright posture on Earth, observations of how spaceflight affects the spine should also provide new and potentially important information on spine biomechanics that benefit the general population.

## PURPOSE:

This study compares quantitative measures of lumbar spine anatomy, health, and bio-mechanics in astronauts before and after 6 months of microgravity exposure on board the International Space Station (ISS).

## STUDY DESIGN:

This is a prospective longitudinal study.

## SAMPLE:

Six astronaut crewmember volunteers from the National Aeronautics and Space Administration (NASA) with 6-month missions aboard the ISS comprised our study sample.

## OUTCOME MEASURES:

For *multifidus* and *erector spinae* at L3–L4, measures include cross-sectional area (CSA), functional cross-sectional area (FCSA), and FCSA/CSA. Other measures include supine lumbar lordosis (L1–S1), active (standing) and passive (lying) flexion-extension range of motion (FE ROM) for each lumbar disc segment, disc water content from T2-weighted intensity, Pfirrmann grade, vertebral end plate pathology, and subject-reported incidence of chronic low back pain or disc injuries at 1-year follow-up.

## METHODS:

3T magnetic resonance imaging and dynamic fluoroscopy of the lumbar spine were collected for each subject at two time points: approximately 30 days before launch (pre-flight) and 1 day following 6 months spaceflight on the ISS (post-flight). Outcome measures were compared between time points using paired *t* tests and regression analyses.

## RESULTS:

Supine lumbar lordosis decreased (flattened) by an average of 11% ( $p=.019$ ). Active FE ROM decreased for the middle three lumbar discs (L2–L3:  $-22.1\%$ ,  $p=.049$ ; L3–L4:  $-17.3\%$ ,  $p=.016$ ; L4–L5:  $-30.3\%$ ,  $p=.004$ ). By contrast, no significant passive FE ROM changes in these discs were observed ( $p>.05$ ). Disc water content did not differ systematically from pre- to post-flight. Multifidus and erector spinae changed variably between subjects, with five of six subjects experiencing an average decrease 20% for FCSA and 8%–9% for CSA in both muscles. For all subjects,

changes in multifidus FCSA strongly correlated with changes in lordosis ( $r^2=0.86$ ,  $p=.008$ ) and active FE ROM at L4–L5 ( $r^2=0.94$ ,  $p=.007$ ). Additionally, changes in multifidus FCSA/CSA correlated with changes in lordosis ( $r^2=0.69$ ,  $p=.03$ ). Although multifidus-associated changes in lordosis and ROM were present among all subjects, only those with severe, pre-flight end plate irregularities (two of six subjects) had post-flight lumbar symptoms (including chronic low back pain or disc herniation).

## CONCLUSIONS:

We observed that multifidus atrophy, rather than intervertebral disc swelling, associated strongly with lumbar flattening and increased stiffness. Because these changes have been previously linked with detrimental spine biomechanics and pain in terrestrial populations, when combined with evidence of pre-flight vertebral end plate insufficiency, they may elevate injury risk for astronauts upon return to gravity loading. Our results also have implications for deconditioned spines on Earth. We anticipate that our results will inform new astronaut countermeasures that target the multifidus muscles, and research on the role of muscular stability in relation to chronic low back pain and disc injury. © 2017 Elsevier Inc. All rights reserved.

**Keywords:** Instability, Low back pain, Lumbar spine, Multifidus, Spaceflight, Unloading

## Introduction

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On Earth, the lumbar spine bears the load of the upper body in upright posture and is stabilized by both passive and active systems [1]. Passive postural stability is provided by the osteoligamentous lumbar spine, which includes the intervertebral discs, vertebrae, synovial facet joint cartilage, and ligaments. This passive system serves to constrain motion via a mixture of complex tissue material properties and geometries. By contrast, active postural stability is provided by muscle tendon complexes that generate force, both locally at the vertebral segments and globally [2,3]. Some muscles act primarily to induce gross trunk movements, whereas others act as stabilizers to support posture and prevent excessive or unwanted motions [4]. Harmony between the passive and active stability systems is coordinated by neural control mechanisms ([1]).

Diurnal loading patterns of activity and rest are critical for maintained spine health and function. For instance, as a poro-viscoelastic material, the disc undergoes significant dehydration, height loss, and concomitant decreased bending stiffness after sustained loading [5]. Decreased bending stiffness can lead to increased tissue strains that trigger paraspinal muscle hyperexcitability [6], thereby increasing active stiffness to compensate for deficient passive stiffness. Rest periods allow the disc to osmotically recover water, height, and bio-mechanical properties. Diurnal periods of activity and rest also facilitate transport of nutrients and metabolites to and from cells within the disc matrix and avascular nucleus pulposus.

Microgravity exposure removes physiological diurnal loads from the lumbar spine, which can hypothetically disrupt lumbar stabilization by deconditioning both the passive, active, and neural stabilizing systems. Prolonged microgravity is known to cause global muscle atrophy [7] and bone loss [8,9]. It is not surprising therefore, that spaceflight puts astronauts at risk for low back pain and disc injury. In National Aeronautics and Space Administration (NASA) studies, astronauts experience localized (non-radiating) low back pain during spaceflight (43% incidence [10]) and a 2.8-fold higher prevalence of lumbar disc herniation following spaceflight [11].

Yet, the detrimental mechanisms of microgravity on lumbar health are uncertain. Reports of increased post-flight stature suggest that microgravity causes “spinal lengthening” [12,13] perhaps due to accumulated swelling of unloaded discs [14]. This hypothesis is supported by in vitro microgravity simulations which demonstrate increased disc height and passive spinal bending stiffness [15], and in vivo bed rest studies on Earth [14]. It is tempting to conclude that such changes explain the increased risk of post-flight disc herniation, but space studies to date have not been able to demonstrate lumbar disc height changes following spaceflight of either 8 days [16] or 6 months [17] duration. Therefore, how microgravity adversely affects the human lumbar spine remains undetermined.

To identify back pain and injury mechanisms, we conducted a longitudinal study of six NASA astronaut crewmembers in whom lumbar spine anatomy and biomechanics were quantified before and after 6 months of microgravity exposure on the International Space Station (ISS). Results of this study are valuable not only to inform countermeasures to reduce disc herniation risk in astronauts, but also to provide insights into spine stability that are relevant to improving back health in the general population.

## Methods

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### Subjects

With institutional research board approval, spine imaging and health data were assessed from six NASA astronaut crewmembers at two time points: before launch (“pre”) and 1 day following 6 months’ spaceflight on the ISS (“post”). We followed up with each subject during a mandatory debrief at 1 year following their return to Earth where they self-reported the occurrence and duration of any low back symptoms or injuries within that year. Subjects included one female and five males (ages ranging from 46 to 55 years). No exclusion criteria were applied beyond NASA’s general health and fitness criteria for spaceflight. As such, our sample was uncontrolled for potential comorbidities such as age-related spine degeneration.

### Magnetic resonance imaging

Lumbar spine imaging was performed with subjects lying supine in a 3T scanner with a 4-channel cervical-thoracic-

lumbar coil (Siemens Syngo workstation; software Version B19, Siemens AG Healthcare Sector, Erlangen, Germany). Localizer images, sagittal and axial T2-weighted images (TR/TE, 3,010/92 ms; thickness, 4.0 mm; field of view, 220 cm; matrix, 320×320; NEX, 2; bandwidth per pixel, 252 Hz/Px; and fat saturation), and sagittal T1-weighted images (similar parameters as T2, except TR/TE was 2,100/9.4) were acquired. The additional T2 map multiecho sequence was then performed as a single midline sagittal image (TR/TE, 1,800/18 ms; inter-echo delay, 18 ms; echo-train length, 1; section thickness, 7.0 mm; field of view, 22 cm; matrix, 320×320; NEX, 1; and bandwidth per pixel, 260 Hz/Px). Fat saturation and anterior saturation bands were applied. The scanning time for the T2 map sequence was 5 minutes 30 seconds.

We measured lumbar lordosis (sagittal angle between L1–S1 cranial end plates on the supine magnetic resonance imaging [MRI]), individual disc and vertebral wedging angles in the sagittal plane [17], disc water content (inferred from T2-relaxation maps [18]), Pfirrmann grade (by co-author CWO), the presence or absence of vertebral end plate irregularities (by co-author CWO), cross-sectional area (CSA), functional cross-sectional area (FCSA), and FCSA/CSA as a measure of fat infiltration of lumbar spine extensor muscles (*multifidus* and *erector spinae*) at L3–L4. CSA and FCSA data were averaged across four adjacent images at L3–L4. The L3–L4 level was used for our analysis because it was most reliably captured by manual segmentation: there was occasional, inadequate image quality at other lumbar levels. End plate irregularities were scored based on presence or absence of morphologic defects and underlying bone marrow signal intensity on T1- and T2-weighted MRI images [19]. FCSA was measured by setting a threshold to isolate lean muscle area within the total CSA [20,21]. The interclass correlation for measurement reliability of lumbar lordosis, vertebral and disc wedging, disc water content, and muscle FCSA ranged from 0.89 to 0.99;  $p < .05$ . All MRI measurements were done using OsiriX DICOM viewer (Version 8.0, Pixmeo, Bernex, Switzerland) and ImageJ (National Institutes of Health, Bethesda, MD, USA) software.

## Dynamic fluoroscopy

In separate active (standing) and passive (side lying) postures, we quantified intersegmental flexion-extension range of motion (FE ROM) for each lumbar disc. A 12-inch surgical C-arm Vertebral Motion Analysis system (VMA; Orthokinematics, Inc, Austin, TX, USA) captured fluoroscopy videos to measure intervertebral rotation that was measured with vertebral tracking algorithms (KineGraph VMA). Controlled bending platforms achieved passive and active postures, with each subject's pelvis bolstered to isolate trunk motion. Each subject was guided through a specified range of 70° sagittal and coronal motion, performed at a rate of 5° per second. The measurement reliability of intervertebral rotation from this method has low variability compared with digitized manual techniques with  $\pm 1.53^\circ$  for intra-rater measurements and  $\pm 2.15^\circ$  for inter-rater measurements [22,23].

## Data analysis

All pre-flight variables were tested for normal distribution using a Shapiro-Wilk test. Statistical analyses included paired *t* tests to compare changes in pre- and post-flight variables among subjects, and simple regression analysis to test for relationships between pre- to post-flight changes for separate variables. Significance was defined as  $p < .05$ . Statistical analyses were performed with Stata (Version 13, StataCorp, College Station, TX, USA) software.

## Results

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### Lumbar lordosis

Following spaceflight, lumbar lordosis decreased (flattened) in all six subjects (average change:  $-11.1\%$ ,  $p = .009$ ; [Table](#)). After comparing the sums of lumbar disc wedging (L1–L2 through L5–S1) and lumbar vertebral wedging (L1–L5), separately, we found that neither lumbar disc wedging nor vertebral wedging significantly changed following space-flight, implying that both changed variably in contribution to post-flight decreases in lumbar lordosis. In two of the six subjects, total lumbar vertebral wedging decreased by  $8^\circ$  and  $9^\circ$  ( $-13\%$  and  $-23\%$  of pre-flight lordosis, respectively), indicating that vertebral bodies lost height on the anterior border relative to the posterior border.

## Table.

Summary of pre- and post-flight data

		n	Pre-flight	Post-flight	Mean diff.	Mean % diff.	p-Value
Lumbar lordosis (°)		6	41.9±12.9	37.2±11.0	-4.73	<b>-11.1%</b>	<b>.009</b>
Vertebral wedging (°)		6	4.4±9.8	2.1±11.2	-2.31	-29.0%	.17
Disc wedging (°)		6	37.6±9.8	35.0±11.2	-2.54	-7.5%	.08
Multifidus CSA (mm <sup>2</sup> )		6	1235.7±252.2	1158.1±231.4	-77.7	-6.2%	.16
Multifidus FCSA (mm <sup>2</sup> )		6	1002.5±319.9	847.3±253.1	-155.2	-14.2%	.06
Multifidus FCSA/CSA (%)		6	80.0±13.1	72.6±15.3	-7.4	-9.3%	.07
Erector spinae CSA (mm <sup>2</sup> )		6	5010.7±815.2	4817.9±1026.1	-192.9	-3.9%	.28
Erector spinae FCSA (mm <sup>2</sup> )		6	3903.7±457.6	3486.5±1186.2	-417.2	-11.5%	.18
Erector spinae FCSA/CSA (%)		6	78.5±5.1	71.5±1.2	-6.9	-9.0%	.09
Active FE ROM (°)	L1-L2	4	6.8±4.3	7.1 ±4.8	0.35	7.7%	.73
	L2-L3	5	8.3±4.3	6.9±4.9	-1.42	<b>-22.1%</b>	<b>.049</b>
	L3-L4	5	8.8±4.9	7.6±4.8	-1.27	<b>-17.3%</b>	<b>.016</b>
	L4-L5	5	8.9±3.1	6.3±2.5	-2.65	<b>-30.3%</b>	<b>.004</b>
	L5-S1	4	6.4±1.4	7.0±3.4	0.59	5.3%	.69
Passive FE ROM (°)	L1-L2	2	8.5±5.5	8.3±8.3	-0.25	-17.1%	.46
	L2-L3	5	3.9±1.1	4.2±2.1	0.27	10.5%	.68
	L3-L4	5	7.4±3.8	7.7±3.1	0.21	17.7%	.56

		n	Pre-flight	Post-flight	Mean diff.	Mean % diff.	p-Value
Disc water content (mean T2 intensity)	L4-L5	5	9.0±2.5	10.8±2.2	1.76	35.7%	.79
	L5-S1	5	11.8±6.0	7.2±4.5	-4.51	<b>-40.0%</b>	<b>.031</b>
	L1-L2	6	109.7±49.9	107.9±55.7	-1.83	-1.6%	.48
	L2-L3	6	93.2±38.1	84.9±36.2	-8.30	-8.9%	.30
	L3-L4	6	71.6±37.1	66.8±29.1	-4.80	-6.7%	.19
	L4-L5	6	78.0±33.5	78.5±22.8	0.49	0.6%	.53
	L5-S1	6	46.7±15.6	51.7±16.2	5.03	10.7	.87
	L1-S1	6	399.1 ±138.4	389.7±127.0	-9.41	-2.4%	.43

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CSA, cross-sectional area; FCSA, functional cross-sectional area; FE ROM, flexion-extension range of motion; SD, standard deviation.

Notes: Includes mean±SD, mean differences, and mean percent differences between pre- and post-flight data, for individual subjects. n represents the number of subjects with pre- and post-flight data available for the analysis. ROM data are available for only five of six crew and in a few cases is missing from L1 to L2 or L5 to S1 because it was outside the field of view.

p-Values are from one-tailed paired *t* tests. Bolded text indicates statistical significance.

## Intersegmental range of motion

Active (standing) FE ROM decreased for the middle three lumbar discs (L2–L3: -22.1%, *p*=.049; L3–L4: -17.3%, *p*=.016; L4–L5: -30.3%, *p*=.004; [Table](#)). Passive (side lying) FE ROM did not demonstrate similar decreases for those middle three lumbar discs, but did show a surprising decrease in motion at the L5–S1 disc (-40.0%, *p*=.03; [Table](#)).

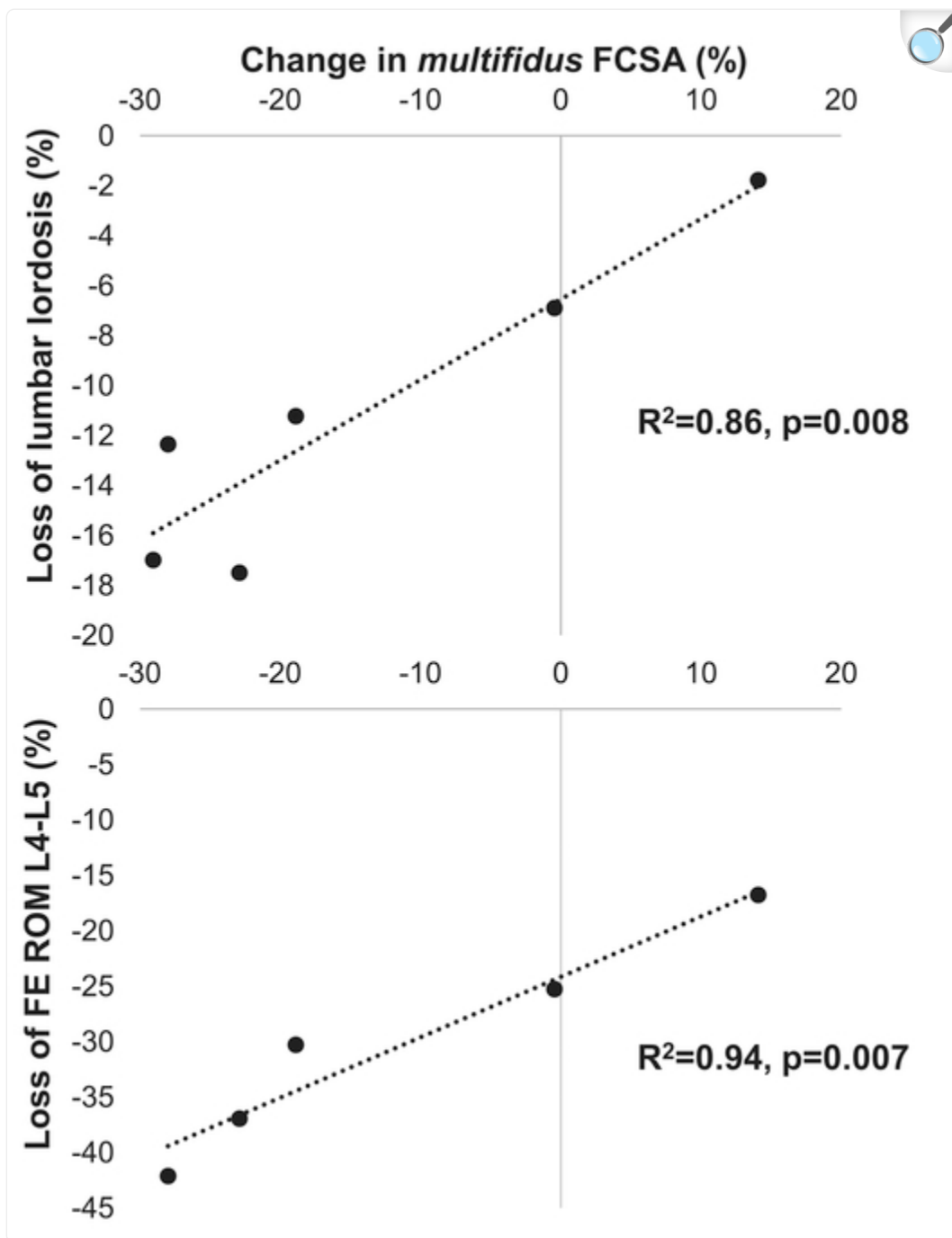
## Muscle atrophy

Cross-sectional area, FCSA, and FCSA/CSA of the lumbar extensor muscles (multifidus and erector spinae) changed variably among subjects, with one subject showing an unexpected increase in CSA and FCSA following spaceflight.



The remaining five subjects experienced a 20% average decrease in FCSA and 8%–9% average decrease in CSA for both multifidus and erector spinae following spaceflight, but there was not a significant decrease in CSA and FCSA for either muscle when accounting for all six subjects ([Table](#)). Changes in multifidus FCSA strongly correlated with changes in the observed lumbar lordosis seen with lying supine ( $r^2=0.86$ ,  $p=.008$ , [Fig. 1](#)) and active FE ROM at L4–L5 ( $r^2=0.94$ ,  $p=.007$ , [Fig. 1](#)). Changes in multifidus FCSA/CSA also correlated with lumbar lordosis ( $r^2=0.69$ ,  $p=.03$ ). In comparison, changes in erector spinae did not relate to changes in supine lumbar lordosis ( $r^2=0.35$ ,  $p=.22$ ), active FE ROM at L4–L5 ( $r^2=0.21$ ,  $p=.44$ ), or FE ROM at any other level affected by spaceflight.

Fig. 1.



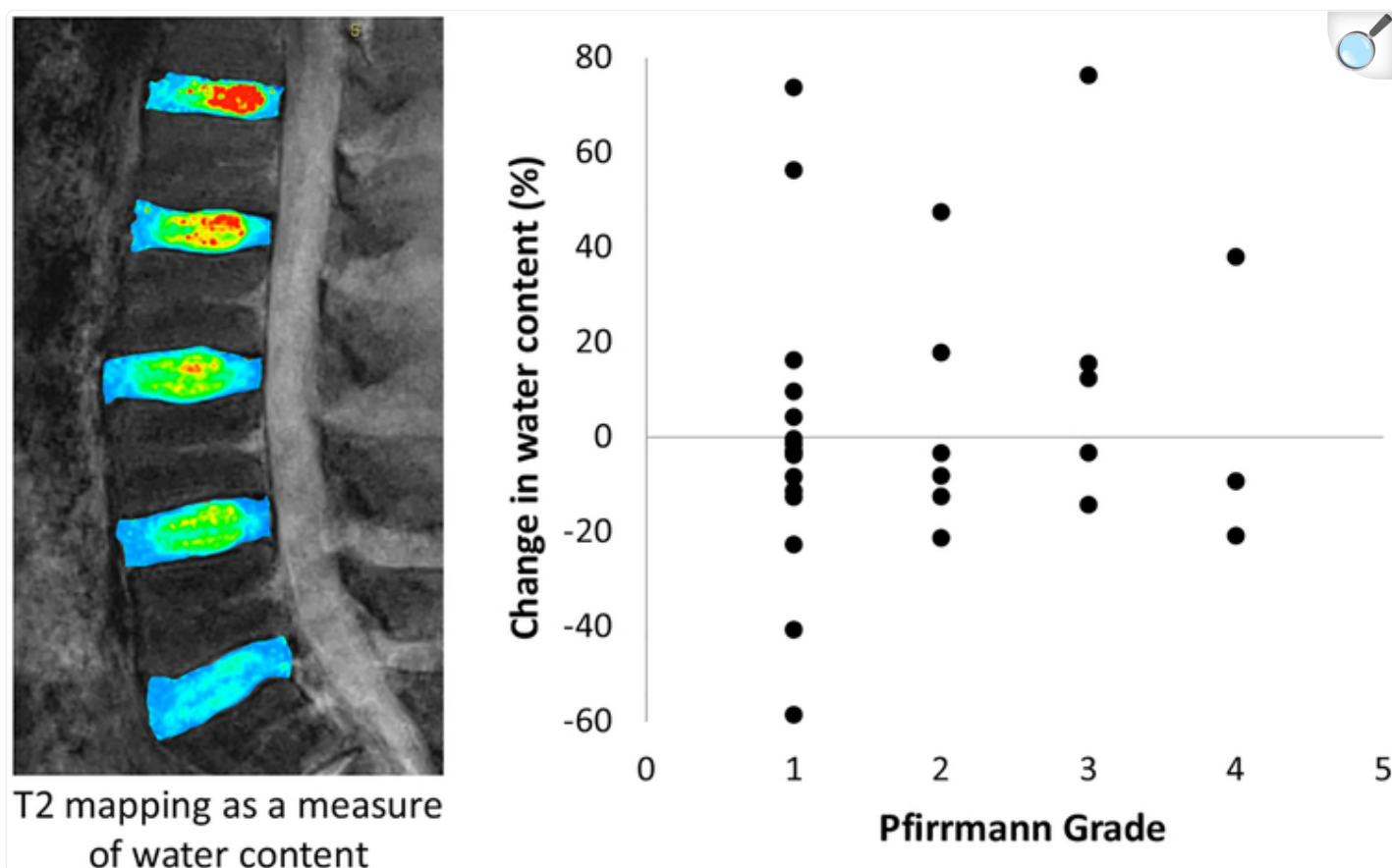
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Scatterplots showing the linear relationship between the change in multifidus FCSA with the change in lumbar lordosis measured while lying supine (Top) and with the change in active FE ROM at L4–L5 (Bottom). FCSA, functional cross-sectional area; FE ROM, flexion-extension range of motion.

## Disc swelling

Disc water content did not differ systematically from pre- to post-flight. This result was true when testing each lumbar level separately and when testing the total lumbar disc water content for each subject ( $n=6$  and  $p>.05$  for all; [Table](#)). We also found no effect of microgravity on water content after pooling all the lumbar discs among six subjects ( $n=30$ ;  $p=.51$ ) and testing whether Pfirrmann grade had an effect on pre- to post-flight change in water content ( $p=.072$ , [Fig. 2](#)). Pfirrmann grade, which is in part defined by disc water content, did not change for any lumbar disc during spaceflight.

Fig. 2.



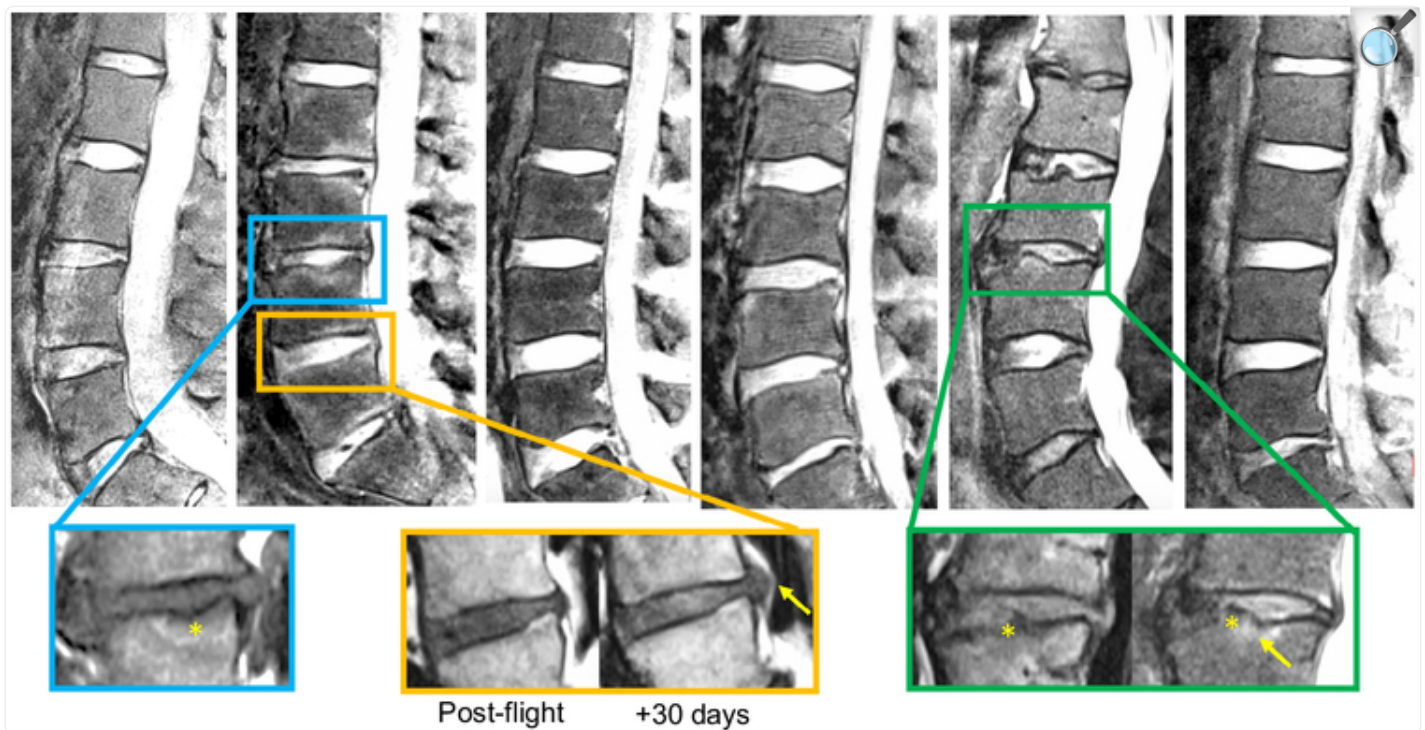
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Change in water content is represented by the % change in T2 mean between pre- and post-flight. We found no systematic relationship between prolonged microgravity and disc water content, with or without adjusting for pre-flight Pfirrmann grade.

## End plate irregularities and post-flight symptoms

We did not find any pre-existing spinal pathologies or Pfirrmann grade to change following spaceflight. Preexisting spinal pathology included only two subjects with end plate irregularities accompanied by positive bone marrow lesions indicative of Type 2 Modic changes ([Fig. 3](#)), a condition previously linked to discogenic pain [[24](#)]. At a 1-year post-flight follow-up meeting with each astronaut, we learned whether they experienced chronic low back pain or injury following spaceflight. The same two subjects with end plate irregularities were the only individuals to present post-flight symptoms, one reporting chronic low back pain and the other reporting both chronic low back pain and a disc herniation at L4–L5 ([Fig. 3](#)).

Fig. 3.



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Mid-sagittal views of T2 weighted 3T lumbar spine MRIs for all six subjects (taken at pre-flight). Example sites for injury and potential sources of pain are pointed out in additional T1-weighted sagittal images (A–C) from two of the subjects who presented post-flight symptoms: (A) indent end plate defect indicated with a yellow asterisk at the cranial L4 end plate, (B) images of post-flight (left) and 30 days recovery following post-flight (right), demonstrating a posterolateral disc herniation indicated with a yellow arrow, (C) T1- (left) and T2-weighted (right) images demonstrated a type 2 Modic change with severe end plate defect (Modic change indicated with a yellow arrow and end plate defect indicated with a yellow asterisk).

## Discussion

This unique longitudinal study underscores the coupled role of passive and active spine stabilizers in maintaining lumbar spine health. We observed that 6 months of space-flight produced significant effects on lumbar biomechanics—the supine lordotic curvature flattened, and the intersegmental ROM decreased. These changes can be detrimental by increasing loads and stresses within the intervertebral discs and vertebral bodies [25,26], particularly when re-introduced to gravity upon return to Earth. However, the mechanisms for these post-flight changes surprised us. Our

results show that the effect of microgravity on active postural stabilizers (eg, muscle atrophy) may be responsible for post-flight increases in stature and risk of disc herniation, rather than the passive postural stabilizers (eg, disc swelling) as previously hypothesized.

We observed that paraspinal muscle atrophy, specifically of the multifidus, was strongly associated with post-flight decreases in lumbar lordosis and intersegmental ROM. The multifidus attaches directly to the lumbar vertebra and acts locally to normally provide the greatest active stiffness in both the sagittal and frontal planes [27–29]. It also has an important role in proprioception and facilitating accurate spine positioning [30]. In this manner, the multifidus acts as a bowstring to accomplish fine adjustment and support of lumbar lordosis [31,32]. Multifidus atrophy in the general population has been linked to chronic low back pain, likely due to alterations in lumbar posture and kinematics [33]. For example, paraspinal muscle atrophy associates with a loss of lordosis [34,35] in patients with degenerative flat back syndrome [36]. The relevance of this mechanism to spaceflight has been previously suggested based on human volunteer studies of prolonged bed rest where multifidus atrophy correlated with loss of lumbar lordosis (approximately 1.5 degrees at L4–L5 [37]). A recent study reported trunk muscle cross-sectional area measured in a single astronaut following 6 months space-flight (not a subject of this current study) and showed that multifidus in the lower lumbar spine decreased more than other trunk muscles [38]. Our current study is the first to demonstrate the strong association between multifidus quality, lumbar lordosis, and intersegmental ROM in vivo.

Microgravity-induced lumbar spine flattening has been hypothesized based on external postural changes (“spinal lengthening”) noted in-flight [12]. Our results indicate that lumbar flattening persists following return to Earth and is linked to multifidus atrophy rather than disc swelling. Lumbar lordosis is a morphologic and biomechanical adaptation particular to humans, enabling effective upright posture and efficient bipedal locomotion under gravitational load [16,39]. Lordosis decreases the maximum forces on spinal tissues by distributing load between the disc and facet joints [40–42]. Muscular support actively stabilizes lumbar lordosis while bearing compressive loads by directing axial load along the lordotic alignment [43]. Therefore, it is not surprising that prolonged removal of gravitational loading can adversely affect lumbar lordosis. Reduced lumbar lordosis driven by muscular atrophy may lead to spinal lengthening via postural changes, as well as increased herniation risk due to increased compressive disc loads and potential for exceeding ROM in flexion [25]. Although our work is limited because we do not have precise pre- and post-flight stature measurements, future work will investigate the effect of microgravity on lumbar lordosis in standing posture and overall sagittal balance.

Our observation that microgravity primarily influences active spine stabilizers is further supported by comparing intersegmental ROM measured in the standing posture (when trunk muscles are active) versus side-lying posture (when trunk muscles are relaxed). Post-flight sagittal ROM of the middle three lumbar segments (L2–L3, L3–L4, L4–L5) was decreased during standing, but not when lying. These data, plus hydration information available from T2-mapping, indicate that disc swelling does not systematically affect post-flight lumbar spine stiffening, and presumably, post-flight disc herniation risk.

Absence of systematic disc hydration increases in our subjects is counter to prior studies where it has long been hypothesized that post-flight changes in astronaut stature arise from supraphysiological disc swelling [12–14]. Our current results, along with recently published data from our team showing negligible changes in disc height [20], demonstrate that disc swelling does not drive changes in post-flight lumbar biomechanics. Prior work on mice demonstrate a degradation of disc biomechanical and biochemical properties following 2 weeks' spaceflight [44–48], but only in the caudal (tail) segments, which may be deteriorating due to excessive use as a means for rodents to propel themselves around the in-flight cages. Furthermore, in prior work, we compared the biomechanical swelling properties of both lumbar and caudal murine discs and report that only the caudal segments showed degraded biomechanical properties following spaceflight [47].

On the other hand, bed rest studies do show increases in disc size following extended inactivity [49,50], an effect that can persist for up to 2 years [51]. An early study by LeBlanc et al. [50] directly compared the effect of 5 weeks of bed rest and 8 days of spaceflight on disc height. They found that bed rest had an effect, but short duration spaceflight did not. The fact that we did not observe systematic lumbar disc swelling from prolonged microgravity may be due to in-flight exercise protocols that axially load the spine. For instance, exercises using the Advanced Resistive Exercise Device (ARED) help maintain spine and hip bone mass [52]. Yet, although exercise protocols designed for ISS are effective for reducing the in-flight loss of general skeletal muscle mass [53], our data indicate they are not preserving core trunk stabilizers, specifically the multifidus. In addition, ground-based studies of ISS countermeasures on individuals following bed-rest induced atrophy show an insufficient effect on lumbopelvic musculoskeletal recovery [54]. This is not surprising because multifidus atrophy can exist in elite, highly conditioned athletes [55], and given the unique role of the multifidus as a stabilizer to stiffen the trunk rather than create motion, it requires specific training regimens directed toward proper activation [56–58].

Although our results show that spaceflight affects the multifidus for all subjects, we found that the risk for post-flight symptoms may relate to pre-existing spinal pathology. Two of the six astronauts presented post-flight symptoms (Fig. 3) and these two subjects also presented end plate irregularities with type 2 Modic changes in the adjacent vertebral bone marrow. End plate irregularities [24,59] and Modic changes [19] have been linked to chronic low back pain in the general population as they represent regions of structural weakening and pro-inflammatory communication between the disc and bone marrow [60]. Weakening at the disc-vertebra junction could be exacerbated by microgravity-induced changes in vertebral bone quality [48,61] and increased disc loading from lumbar flattening [25] that together heighten risk of failure and injury at the disc-bone interface when reintroducing a relatively less stable lumbar spine to gravitational load.

Like many prior studies on subjects exposed to microgravity, this work is subject to limitations in sample size and lack of in-flight data. More work needs to be done to understand whether or not there is significant disc swelling during spaceflight. Although our work indicates that disc swelling is not significantly different following spaceflight, we acknowledge that a considerable amount of changes in disc water content could happen in the ~24-hour period between



landing and post-flight imaging. Further work is being done using ultrasound to address disc height during spaceflight.

Regardless, our results demonstrate that in spite of negligible changes in disc swelling, significant postural and stiffness changes occurred following spaceflight and related to changes in multifidus muscular stability. Future work involves collecting similar data on additional astronauts and aims to implement countermeasure exercises targeting the multifidus. If multifidus health is maintained during spaceflight, astronauts with pre-existing spinal pathology may be less vulnerable to post-flight symptoms and injury.

This prospective study sheds light on mechanisms of post-flight back pain and disc injury—multifidus atrophy appears associated with spinal flattening and increased stiffness. We hypothesize that these factors, when combined, increase injury potential, particularly in those subjects with pre-existing evidence of vertebral end plate insufficiency. These results have implications also for deconditioned spines on Earth. We intend to use these results to develop countermeasures targeting the multifidus muscles and new research on the role of muscular stability in relation to chronic low back pain and disc injury.

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## Footnotes

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FDA device/drug status: Not applicable.

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